THE QUESTION OF OPERATION FOR NON-PENETRATING INTRACRANIAL TRAUMA.*

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NON-PENETRATING injuries of the cranial contents may be divided into four classes in their consideration from a therapeutical standpoint. First—Those in which the injury is so slight that recovery is certain without operation. Second—Those in which the severity of the injury is so great that death is inevitable in a short time. Third—Those in which the injury is of such a nature that operation is positively indicated. And fourth—Those in which the indications for and against operation are more or less evenly balanced, the border-line cases. The first and second classes need but a brief consideration. In the first are included cases of mild or moderate concussion without gross anatomical lesions. The diagnosis of this condition usually offers no especial difficulty. As a rule, the inflicting violence is not very great. The patient loses, to a greater or less extent, consciousness; the face is pale, there is vomiting, muscular relaxation, including the sphincters, and a loss of tone in the blood vessels, causing sweating and feeble rapid pulse. The reflexes are usually at first diminished, and then exaggerated. The pupils are, as a rule, dilated. The respirations are shallow and rapid. The temperature is not elevated, and may be lower than normal. The course of the condition is toward recovery, and in a short time, with rest in bed, the administration of stimulating enemata, cardiac restoratives, and external heat, a marked improvement is noted. It must be remembered that this picture of concussion may be present as a complication in any brain injury, and may mask the symptoms of a more serious lesion. The more severe cases of concussion belong to class four, and will be considered there.

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Under the second class belong those cases in which the damage to the brain is very extensive, or implicates the centres of the vital functions in the medulla. Extensive damage (of this sort) results usually from multiple fractures with large intracranial hemorrhage and laceration of brain tissue. The latter are occasionally seen without fracture. A localized injury involving only the medulla is, fortunately, rare, for death is almost instantaneous. The patients of this class present such a varied symptomatology, and their condition is one of such extreme shock, that no accurate diagnosis can be made. Death usually ensues within a few hours, and no treatment is indicated, other than the means usually employed to combat the shock. This, however, should be done energetically and consistently, because it occasionally happens, that the brain damage is not so great as the shock indicates, and recovery is possible.

The class of cases in which operation is plainly indicated, and that class where its value is doubtful, are of especial interest. In deciding for operative interference in any case of intracranial injury, certain well founded principles have been formulated in the past, and our present knowledge in no way alters them. First—An accurate diagnosis of the pathological lesion existing must be possible. Second—The possibility of relieving this condition must be present. Third—The operative violence in accomplishing this must not be excessive. And fourth—It must be reasonably certain that complete recovery will not take place without operation.

Cerebral localization has made great advances through the work of the physiologists, neurologists and pathologists during the last decade, and accurate diagnosis is accordingly advanced. In dealing with a trauma, however, considerations arise which are not present in brain tumor and abscess, namely: those due to the general concussion and to the possibility of multiple brain injuries being present. For these reasons, the advances in localization are not of so great value in working out the problems presented by our subject, as they are in a more circumscribed pathological lesion.

A careful analysis of all non-penetrating intracranial injuries shows that only two sub-groups can be properly made under classification three, where operation is positively indicated. The first of these is when the brain injury is due to a direct damage to the brain by a depressed fracture, and the second, the classical middle meningeal hemorrhage, or hemorrhage from a cerebral vessel directly affecting a sensorimotor area. In the first sub-group the procedure of dealing with the fracture and the underlying brain injury is well established along definite lines, and needs no elaboration at this time. Emphasis must, however, be laid on the necessity of exploring every suspected fracture of the calvarium, even though no evidence of actual brain injury is present, for such injury may exist in the absence of symptoms and late epilepsy often supervenes on such a condition.

The clinical picture in the second sub-group is usually well defined, and rarely is a diagnosis not made. The history of the injury, followed, usually though not invariably, by longer or shorter periods of concussion, then freedom from symptoms, with the later onset of compression, muscular contraction and paresis on the opposite side, are too well known to require any comment. Operative interference in all cases of these sub-groups is positively indicated. The following case of Dr. Gallaudet, by whose courtesy I include it, is an excellent illustration.

Case I.—Admitted to Bellevue Hospital on November 27, 1907, in the service of Dr. Gallaudet, with the history of having received a fist blow over the left side of the head the day before. He was not rendered unconscious, but almost immediately became aphasic. On admission he was aphasic, and showed a right-sided paralysis of the arm and face. Operation showed a stellate fracture with extra and subdural hemorrhage, as well as hemorrhage in the brain cortex over the area indicated by the symptoms. Recovery.[1]

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[1] For a full report of this case by Dr. Gallaudet, see page 122.

The following cases, however, show the difficulties which may arise, and the caution which is needed in drawing conclusions.

CASE II.—*Trauma over left parietal region from falling timber. Subdural hemorrhage over right precentral convolution with late development of irritation and pressure symptoms. Operation with recovery.*

A man, aged 49 years, colored, was admitted to Lincoln Hospital on Feb. 27, 1906, with the following history: On Feb. 21st he was struck on the head by a large piece of timber falling from a height of one or two stories. He was knocked to the ground, was picked up unconscious and taken to a hospital in an ambulance. He regained consciousness in about twenty minutes, but was excitable and irritable. There was a large scalp wound over the left parieto-temporal region. This was sutured. He continued in his irritable and rather unmanageable condition during the next six days, without, however, manifesting any local cerebral symptoms. In addition to his irritability, he had several attacks of vomiting. He was taken home against advice. On his admission to Lincoln Hospital, six days after the injury, the following notes were made: Patient has a healed scar on the left side of his head, six inches long and curved like an operative incision. Patient seems drowsy, yet at intervals he is irritable, restless and unmanageable, trying to get out of bed. Patient does not respond to questions, and on being aroused looks at one with a vacant expression. Heart, lungs and abdomen all normal. Extremities: no change in sensations apparent. No paresis or paralysis. Reflexes: markedly increased. Control of bladder and rectum perfect. No change in pupils. Shortly after admission patient had a convulsion, which was reported by the attendant to be general in character.

During the following three days the convulsions were repeated several times, were of very short duration, and no evidence that they were at all localized could be obtained. On March 2nd these convulsions began to occur with great frequency, about every twenty minutes, for periods of an hour at a time, followed by a period of rest. They were becoming longer in duration, and the following notes on their character were then made: There was a vacant staring expression of the eyes, with a gradual conjugate deviation toward the left; after four or five seconds, there was a tonic contraction of the muscles of the left side of the face, then a drawing of the head downward on the left shoulder, with an elevation of the latter; then tonic contractions in the arm, and then in the leg of the left side. This phase occupied about fifteen seconds, and was followed by clonic spasms of the same parts and in the same order. The whole convulsion lasted from sixty to ninety seconds, during which time the patient was totally unconscious. He then gradually regained consciousness and the contractions ceased. At this time, when he had altogether about twelve convulsions, he for the first time showed a decided left facial paralysis, and marked weakness of left arm and leg. This was on the ninth day after the injury, the first convulsion having been noted on the seventh. On the eighth day his condition had apparently improved. Operation was performed on March 2nd, nine days after the injury, and as soon as the localized character of the convulsion and the paralysis was evident. Under ether, a curved incision was made just above the right temporal ridge, about eight inches in length, and carried directly down to the skull, the flap being turned downward. The fissure of Rolando was now marked out, and a trephine opening was made over the face centre, in ascending frontal convolution, and enlarged upward with the rongeur, to an area of about two inches in diameter. The dura showed no pulsation, and a clot could be seen beneath it. Dura was divided around the line of the bone opening, and a large organized clot was removed from the cerebral cortex. Electrodes applied to the facial centre produced a prompt contraction of the muscles of the face on the left side. No response could be obtained in either of the extremities, possibly because the nerve cells here were too much damaged, and inspection indicated that the cells for the arm were more damaged than those for the face. The opening in the skull was not extensive enough to reach the leg centre. The dura mater was closed with catgut sutures, a small rubber tissue drain put down through it, and the scalp sutured back in place. It was noted that the scalp was quite œdematous, probably due to lymphangitis following the original scalp wound.

Post-operative notes: March 4th—Recovered from anesthetic without incident. Slightly excited during first twelve hours, and then became rational, but continues rather stuporous. Can be roused and gives correct account of injury, and other details of his residence, work, etc. Still has considerable weakness of left upper extremity, and less of the lower extremity. Is able to make coordinated movements. Slight spasticity of left lower extremity, none of upper. Left-sided facial paralysis, and left deviation of tongue. Upper branch of facial less paralyzed than lower. Examination of reflexes unsatisfactory. No oculo-motor paralysis. March 6th—Patient less rational than formerly. More stuporous. Complains of pressure on head. Very restless. Left arm can be moved only with great difficulty. Face more markedly paralyzed. Wound examined and pus found along the suture line. Considerable distension. Opened after stitches were removed. Infection all through the scalp, due to previous lymphangitis. Pus infiltrating tissues down to the dura. Dura thickened, and brain apparently well walled off by dense adhesions. Wound area opened up widely and dressed with free drainage and bichloride solution. March 9th—Wound very much cleaner. Both sides granulating well. In centre, still suppurating. Left arm can be moved more freely. March 11th—Wound granulating well. Patient more rational. Movements on left side stronger. March 13th—Patient improves slowly. Mind brighter. Movements of left side gradually returning. Can put left hand to mouth and nose with effort. Cannot hold up a single finger of his left hand. March 15th—Wound granulating well. Pulsations fair. Complains of being in bed. Left facial paralysis less marked. Can nearly hold up finger of left hand. March 27th—Wound granulating well, nearly free from pus. Mental condition practically normal. Left leg can be used nearly as well as the right. Left arm not so powerful as right. Power of coordinated movements not entirely regained. With eyes closed, fingers do not meet by several inches. Left not so strong as right. Facial paralysis still slightly present on left side of face. Left eye cannot be shut tight. Left angle of mouth can be drawn back but a very little. April 10th—Patient discharged, forty days after the operation. Wounds entirely closed. Still showed slight incoordination of left side of body, and slight left-sided facial paralysis. He walks without any dragging of the foot. Mentally, he is normal.

April 25th—Eight weeks after his operation there was absolutely no evidence of his injury remaining except the scars on his head, and the brain pulsation beneath the opening in the skull. There is no evidence of any irritability of the brain due to adherent dura. The patient was again seen on February 10th, 1908. The only evidence of his trouble is found in a loss of general strength and an irritability of temper. No paralysis or convulsions have developed.

The interesting point here was the late onset of localizing symptoms. He had been under observation in two hospitals for nine days, before any evidence on which to base an accurate diagnosis was available. And yet at operation a subdural hemorrhage was found directly over the motor area, on the side opposite to the injury. A case reported by Dr. Krauss, *Amer. Journal Medical Science,* Vol. 128, is the almost exact counterpart of this.

CASE III.—*Trauma over right parietal region from falling brick. Paralysis on right side. Operation on left side. Extra and subdural hemorrhage over right motor area. Death.*

Man, age about 30 years, was admitted to Lincoln Hospital May 19, 1906, with a history of having been struck on the right side of the head by a falling brick. Patient was rendered entirely unconscious and remained so on his arrival at the hospital in the ambulance. Examination at that time showed the following conditions: Patient entirely unconscious. Cannot be aroused. Pupils equally dilated. Do not react to light. No strabismus. No facial paralysis. Hematoma on right side of head over the posterior frontal area. Suggestion of a depressed fracture of the skull, but not definitely determined. No laceration of scalp. No bleeding from ears, nose, or mouth. Spine apparently intact. Chest normal. Pulse very slow, though regular. Second sound of the heart good. Pulse shows slight increase in tension. Lungs normal. Breathing varies. At times, Cheyne-Stokes in character, at other times it became stertorous. Abdomen normal. All superficial reflexes are delayed, though present. Knee-jerks present but not exaggerated. Cremasterics delayed. All extremities moved normally though hard to elicit on account of unconscious state. Sensations delayed but present. One hour later an incision 2½ inches long was made on the right side of the head, through hematoma down to the skull. No fracture discovered over the parietal bone anywhere on the right side as far as felt. One hour later, patient still entirely unconscious. Color good. Pulse slow but of good volume. Slight increase in tension. Breathing more regular and deep. Pupils: left slightly contracted, reacts to light; right dilated and does not react. *Right eyelid* *partially paralyzed with a tendency to right facial paralysis.* Irritation with pin causes no response on right side. On left side of face causes shutting of eyelids and twitching. *Right arm and leg partially paralyzed,* though not entirely so. Some response to pin. Toes moved some. Knee-jerks absent on right side. Very slight on left. Cremasteries delayed on right side. Present on left.

Three hours later. Patient entirely unconscious. Supraorbital pressure causes facial movement on the *left side.* Only slight on *the right.* Pupils: right dilated, left contracted. Neither reacts to light. No strabismus. No nystagmus. Pin pricking on whole of left side, including face and extremities, causes active movements on that side. Right side pricking causes movements of left extremities, with evidence of sensations but feeble movements of right arm, face, and lower extremity, those of the right thigh being a little stronger than those of the leg. Right knee-jerk exaggerated. Superficial reflexes of right side about normal. Knee-jerk of left side about normal. Superficial reflexes on left exaggerated. While eliciting cremasteric reflexes, patient voided urine and was apparently conscious of the act. Pulse still slow. Respirations labored and stertorous. Five hours after the injury patient was etherized. Head cleansed on table and a curved incision was made over left temporal region about in line with the temporal ridge. Incision down through all tissues to bony skull. Flap raised intact, and bleeding vessels caught and clamped. Flap turned down and covered with hot towel. Surface of skull exposed over left motor area (previous to incision by mensuration the motor area of left hemisphere was determined and the skull marked for arm area). Skull trephined. The dura appeared dark, but seemed to pulsate. With continuous saline irrigations trephine opening was then enlarged up and down, and to both sides, exposing an area about 1½ inches wide and 2 inches long. Dura rather dark, but brain pulsating. Large congested vessels in the dura. Incision longitudinal in character made through the dura and enlarged with scissors. Vessels of the pia mater exposed, greatly congested. No subdural hemorrhage. Brain tissue itself rather darker than normal, and under severe pressure. About the margins of the opening, brain appears better and more normal in color. Crucial incision now made in the dura, and brain allowed to bulge out fully half an inch.

Immediately, patient's breathing, which had been labored and stertorous previously, became quieter and deeper. Pulse found now to be increased in frequency, and more nearly normal rate. Character of brain area exposed, changed when left to bulge for a time. Central area in opening has several dark spots, as if intracerebral hemorrhage had taken place. Surface about margins became nearly normal in character. Bulging seemed to relieve some of the intracranial pressure. Puncture made through pia mater, but no fluid obtained. Slight venous oozing followed. Cerebral puncture disclosed no deep hemorrhage. Flap brought back, and with dura left open the entire tissues of the scalp were sewed back in place with continuous catgut suture. Rubber tissue drains inserted, and sterile dressing applied. Patient returned to ward and put in bed with head raised. Patient passed comfortable night. Still unconscious, though not deeply so. Breathing quietly. Pulse a little rapid. Slight movement present in right arm. Left hand had to be tied to keep him from tearing off dressing. Face flushed. Urine passed involuntarily in the bed. Slept part of the night.

May 20th.—9.30 A.M. Patient seems somewhat conscious. When name is spoken at times opens one eye and appears to hear and understand. 2 P.M. Face flushed. Breathing quietly. Pulse good with less tension. General condition remarkably good. Right pupil dilated, left contracted. Both react to light, though right, very little and slowly. Motor system same as yesterday previous to operation. Left side moved normally. Right leg, thigh and toes all moved slightly, thigh the most. The only difference is in the upper extremity, which seems more paralyzed than yesterday. To stimulation, only movement of arm elicited. Forearm and hand completely paralyzed. Right eyelid and face moved more than yesterday, though still somewhat paralyzed. Sensory, same response as yesterday. Heat, cold and pain tried. Apparently not so sensitive to heat and cold on left side as to pain. Right side not distinguishable. Right knee-jerk exaggerated. Left normal. Cremasterics, right not obtained, left normal. Superficial abdominal delayed on right, exaggerated on left. Bowels have not moved as yet. Active incontinence of urine. Mental condition improved. Patient seems to know his name and resists when catheterized. When pricked with pin, seizes hand pricking him with his left hand and mutters incoherently. May 22nd—General condition not so good. Mentally, more deeply unconscious. Left pupil dilated and does not react. Entirely paralyzed on right side. No response to pin pricks. Right knee-jerk slightly exaggerated, but not markedly so. At times has to be catheterized, at others has active incontinence. Deep pricking on right side of body and extremities produces movements of the other side of body. Not quite as marked as when the same prick is applied to left side. Knee-jerk right side lost. Cremasteric present. Left pupil reacts. Right pupil reacts slowly. Right axillary temperature 102.2 F. Left axillary temperature 102.6 F. May 23rd—Patient responds to name, and when asked questions mutters incoherently, though he seems to hear and partly understand. Paralysis on right side total. Sensation on right side dulled. Pin pricks cause very little movement on the opposite side. Left side sensations less marked. Left pupil contracted and reacts. Right dilated, and it also responds slowly. Patient breathes rather heavily and seems comatose most of the time. Urine passed involuntarily. Bowels have moved only with enemas. Sphincter control seems perfect. Reflexes: right knee-jerk lost, left present. Cremasterics present. These findings remained unchanged until death on May 25th.

Autopsy.—On removing skull-cap considerable amount of extradural soft jelly-like blood clot was found over the *right motor* area and temporosphenoidal lobe. None on the left side. On opening dura an extensive clot was found covering the lower half of the right ascending frontal convolution extending forward toward the frontal lobes. Just above Sylvian fissure, in front of Rolandic area, the clot was rather firm and the brain surface showed considerable pressure damage. Over operative wound, brain seemed entirely normal. Whole left motor area was entirely free from clot or evidence of injury, either extra or intradural. A small hemorrhage was found deep in the brain at the site of one of the needle punctures. No fracture of the skull found. Examination of the medulla showed a normal crossing of the pyramids.

This case illustrates a condition not infrequently observed, namely, the brain injury on the same side as the paralysis. Three explanations are given: first—a rare condition of uncrossed pyramids; second—a counter pressure on the opposite side from the hemorrhage against the bony wall, producing localized pressure there which is not felt immediately beneath the soft cushion of the blood; and third—an error in observation, that is, the voluntary paralysis exists on the side which is moved when irritated, the movement being reflex; whereas, on the non-paralyzed side, no movement takes place, because the reflexes are inhibited by the normal cerebral influence. The latter seems the more plausible, though in this case the movements appeared to be voluntary in character. After the operation, his efforts to remove the dressings certainly could not be called reflex, and they were made powerfully with the left arm—that is, on the side where the brain damage was subsequently found—so that the arm had to be tied to the bed. Whatever the explanation is, the therapeutic procedure should be to open both sides of the skull in such cases. The neglect to do so here probably cost the patient his life. The muscular reflexes, as usually elicited, it will be noted, varied from time to time, sometimes being greater on the right side, and sometimes on the left. The variations in the pupil, too, were irregular and not to be classified, though as a rule they were of the Hutchinson type described below. These curious variations in reflexes are those reported by all observers, and emphasize the fact that clinically the teaching of physiology concerning the reflexes is of little value. Their confused condition in spinal injury has been the subject of much unsatisfactory study. This case is very instructive when considered in conjunction with Case II. The injuries were very similar in their nature, but the results were quite divergent. In Case II the hemorrhage was only subdural on the side opposite to the injury, and the paralysis was contralateral to the hemorrhage and slow in developing. In Case III the hemorrhage was both extra and subdural on the side of the injury, and there was a homolateral paralysis developing early. The late development in Case II seems to have been due to a chemical destruction of nerve cells by the changes in the blood clot, while the early development in Case III was undoubtedly due to the immediate effects of the pressure.

Loeb has shown that the cerebral cortex is not excited by the ordinary chemical stimuli which affect nerve fibres. This does not preclude the possibility, however, of a stimulation due to a change in the cells themselves when subject to a condition such as that found in Case II. No recent hemorrhage was present, and the pressure per se did not seem sufficient to cause the symptoms observed. Some of the cells, too, seemed inexcitable to faradism, which still further points to serious damage in their structure having taken place.

Case IV.—*History of old trauma over left side of forehead. Symptoms of late abscess over left motor area. Operation without finding lesion. Death. Autopsy showed no brain lesion, but a condition of the kidneys suggesting acute uræmia as cause of symptoms and death.*

Man, age 28, admitted to Lincoln Hospital April 6, 1907, in an unconscious state, the following history being obtained from his family. Three years previously the patient was struck on the left side of forehead. Was unconscious from the blow for over twenty minutes. Recovered in a short time with seemingly no ill effects. About six months ago patient suddenly had trouble with his speech. Was unable to speak for a time. Attack similar to "petit mal." Would be all right the next day. Since that time he has never been quite the same. Speech thick and hard to understand. After succeeding attacks he seemed to become depressed in spirit, and at times acted as if he were drunk or drugged. About two months ago he was treated at this hospital for injury to left foot, from stone falling on it, causing a bursting laceration on the sole. Wound cleaned, drained and sutured. Healed up very rapidly. At this time he was considered mentally weak. He was brought to the hospital in an ambulance yesterday, while in an unconscious condition, and having convulsions, general in character. According to his wife, who found him in this condition, he had never had any attacks of this kind before.

Examination showed the patient to be well nourished. He was entirely unconscious, lying in bed with head turned to the left; eyes also drawn to the left. Pupils equal, regular, reacting to light. Eyes oscillate slightly and at times slight incoordination is seen. There is constant twitching of right hand, particularly middle fingers. Also twitching at times of right side of face, right leg, and whole right side of body. When twitching is most marked, left leg becomes involved. The temperature ranges from 100 to 103.5. Pulse 110. Respiration 25 to 30. Leucocytes 20,000. There was no paralysis noted, though the right extremities seemed weaker than the left. The picture here given is that of a late abscess following a trauma, and the greatest point of irritation seems to be over the motor area for the face and arm in the left hemisphere. The conjugate deviation of the eyes is away from the side on which the extremities are involved, and not toward it, as is the rule. One discrepancy is noted. After twenty-four hours' observation, operation was advised and accepted.

Operation, April 6th.—The motor area of left hemisphere was determined, and the skull marked for arm area. A horseshoe shaped incision was made through all the tissues down to the skull, over left motor area, flap being turned down and protected by a hot towel. Skull trephined and opening enlarged with rongeur. Dura appeared normal through opening about 1½ inches wide and 2 inches long. Incision made in dura exposing cerebrum, just anterior to fissure of Rolando. Brain pulsating normally, no bulging, vessels in pia seemed enlarged, but membranes about them appeared abnormal, some places being pearly white, but mostly yellow. These areas small and opaque. The exposed area of brain was then thoroughly explored with needle in search of abscess, but with no result. Dura closed with catgut sutures. Flap sewed back in place with interrupted and continuous catgut sutures. Dressing applied. April 7th—Patient still in unconscious condition. Seemingly a little brighter. Pupils equal, dilated. Some drooping of left eyelid. Slight nystagmus. Eyes still drawn to left side. Pupils react to light, but sluggishly. Marked flattening of right side of face. Right arm flaccid with some twitching of hand, but not so continuous. Left arm rigid. No reaction to sensation or pain on either side. Right leg flaccid; purpuric condition on leg extending from ankle to upper third of thigh. No reaction to pain or sensation. Knee-jerk increased. Babinski present. Left leg rigid. Knee-jerk normal. No Babinski. Cremasteric reflexes absent on right side, present on left. Abdominal reflexes active. Dermographia all over body, marked on abdomen. April 8th—Patient had a general convulsion. Twitching in hand not present. Condition worse. Rise of temperature to 103.5. Very rapid small pulse. Breathing irregular. Has to be fed by catheter through the nose, and per rectum. Urine and feces lost in bed. April 9th—During a convulsion this morning, eyes were first noticed becoming incoordinated and drawn to right side, then twitching seen on right side of face, tetanic movements rapidly increasing, then extending to fingers of right hand and arm, next the right leg was involved, then becoming general. Attack lasted about two minutes, dying out where it began. No increase in knee-jerk on right side. No Babinski. Death sixty hours after operation, with no change in condition last noted.

Autopsy.—The brain exposure was found to be exactly over the face and arm area. Nothing abnormal was found on the surface, either in the dura or the brain itself. Sections showed no abnormality other than those due to the needle puncture. At the end of one of these 1½ inches from the surface, beneath the arm area, there was a clot about ⅜ inch in diameter. Other smaller clots were also found. This is interesting as explaining in part the greater degree of paralysis after the operation. The kidneys showed a condition of acute degeneration which the pathologist reported to be due to a toxemia of some sort. There was no evidence of any chronic lesion. The possibility of the convulsions being uræmic, cannot be excluded, though previous to operation, and following it, urine was freely excreted. It contained considerable albumin and some blood, particularly just prior to death, but this was believed to have been due to his toxic condition. All the other organs were found normal, and no additional cause of death could be determined.

It is well known that uræmia may cause one-sided convulsions simulating a brain irritation. In this case, however, the history of the old head injury, the weakness on the convulsed side and the elevated temperature, with the leucocyte increase, all seemed to justify the suspicion of brain abscess.

CASE V.—*Unconscious alcoholic patient with head contusions. Convulsions on left side. Operation and death. Autopsy showed localized cortical softenings.*

Unknown man, age about 45 years, admitted to Bellevue Hospital September 27, 1906, from Harlem Hospital with a diagnosis of alcoholism. He was in coma most of the time, but soon after his admission it was noticed that he was having frequent general convulsions, one every hour or so. On Sept. 29th the convulsions became localized to the entire left side of body, each one lasting from one half to two minutes. In the intervals the left side seemed to be paralyzed. Complete examination at this time showed the man to be partially conscious, but unable to answer questions. He showed every evidence of being markedly alcoholic. Supraorbital stimulation brought on convulsions of the left side of the body, eyes deviating to the right; pupils are equal and moderately dilated, and react normally. Pulse regular; fair force; moderate tension. Knee-jerks absent on both sides. Left upper and lower extremities paralyzed. Right extremities both moved voluntarily. No paralysis of face, but some convulsive twitching on both sides.

Examination of head shows general contusions and bruises over face and forehead. The case was considered one of probable alcoholic cerebral œdema, but the presence of contusions on the head, and the localized nature of the convulsions and paralysis, led me to operate rather against my judgment. Accordingly the motor area was exposed over the left hemisphere, and the brain found to be in a congested condition, and somewhat œdematous, but no localized process to account for the left-sided convulsions. Following the operation, the patient's condition was practically unchanged, and he died about two hours later.

Autopsy.—The brain was found to be markedly œdematous, with areas of circumscribed softening scattered irregularly over the cortex. This condition was especially marked in the first temporosphenoidal convolution on the right side, and in the angular convolution, both of which are situated well behind and inferior to the motor areas. Operation in this case was done on insufficient data. The element of an alcoholic cerebral œdema, with the localized softening, was given too little weight.

A summary of these four cases shows, one with distinct local hemorrhage over a motor area, giving no symptoms until the ninth day, and three with all the symptoms of a localized motor lesion, in which, at operation and autopsy, no such lesion could be demonstrated. Nothing could better illustrate the difficulties encountered in making an accurate diagnosis in these conditions, and the necessity of being cautious in deciding for operation.

There is left for our consideration the fourth group of our classification, the true border-line cases, and this constitutes one presenting even greater difficulties. A study of the manifold functions of the brain, its liability to serious injury despite its complete protection in the skull, its inaccessibility to the surgeon, and its delicate structures, reveals at once the cause of these difficulties. The cases coming under this group may be conveniently divided pathologically as follows: First—Serious concussion. Second—Contusion, that is, multiple small lacerations of brain tissue and blood vessels. Third—A more extensive laceration confined to a limited area. Fourth—Hemorrhage not giving distinctly localized symptoms, that is, not causing irritation or compression over an area of known and demonstrable function. Fifth—A combination of two or more of the above. in the first sub-group, serious concussions, a careful study of the symptomatology will usually be rewarded by a correct diagnosis. There is, almost without exception, a history of an injury acting rather diffusely over the head, followed by immediate unconsciousness. Vomiting is present, but not of the projectile type seen in compression. The whole appearance is one of extreme shock, or collapse, except that the pulse, while soft and compressible to the vanishing point, is not always correspondingly accelerated. This condition lasts a varying time, and is *usually* followed by slow improvement, if the result of a pure concussion, without gross lesions. Occasionally, however, instead of recovery a new set of symptoms is ushered in. The picture then is more complex, and with the symptoms described above, there are mingled those of cerebral excitation. These are restlessness, irritability and increased reflexes. The patient resents markedly any attempt at an examination. Instead of improvement, he passes from this stage into one of deepening coma, and the slow, full high tension pulse, and slow deep respirations mark the onset of a condition of compression. This may, of course, be due to a hemorrhage, but it also develops without this when the disturbed tone of the blood and lymph vessels allows the transudation of serum to produce a brain œdema. The following case fully illustrates these points.

CASE VI.—*Diffuse head trauma. Concussion with later onset of cerebral irritation symptoms and compression. Decompression operation. Recovery.*

A boy, ten years old, was admitted to Lincoln Hospital February 4th, 1906, at 2 P.M. with the history of having fallen a distance of 20 or 30 feet, and landing on his head and shoulders. No one actually saw him fall, so that it was impossible to get accurate data on the above points. He was brought to the hospital by ambulance in a condition of considerable shock, and partial coma. He could be aroused with difficulty; surface cold and pale; temperature 99.2, pulse 120 and weak, respiration 32. There was no paralysis. Pupils were dilated. No localizing symptoms of any sort could be made out. Examination of the head showed an extensive hematoma over vertex and left parieto-frontal region. No evidence of fracture could be made out under this hematoma. There was no bleeding from the ears, mouth or nose, nor any subconjunctival hemorrhage.

The child was put to bed, and the usual remedies for shock, including rectal irrigations and morphine, were given. In the course of an hour the shock had considerably lessened, and the coma was less deep. He continued to recover from the shock, but the coma again deepened, and the irritability on being aroused was becoming excessive. No coordinated response could be elicited in any way. He resented very markedly any manipulations, or any effort to make him answer questions. He failed to recognize his father. All the reflexes were markedly exaggerated, but no paralysis or anesthesia could be made out. His condition was diagnosed as one of severe cerebral concussion, with progressive changes in the cerebral vessels, and beginning œdema of the brain. Under ether anesthesia, two hours and a half after admission, incisions were made over the hematoma, and the skull explored. No evidence of fracture could be found. It was determined to open the skull for the purpose of exploration and decompression. Accordingly, the temporal muscle on the left side was exposed along its origin, its fascia turned back by a semilunar incision, and the fibres separated vertically, according to the method advocated by Cushing. A one-inch trephine opening was then made at a point one and one-half inches above, and one inch in front of the external auditory meatus, exposing the dura. This was seen to be dark in color, very tense and without pulsation. No extra dural hemorrhage was found. The skull was rongeured away in all directions, making an opening of about 2½ inches in a longitudinal by 2 inches in a vertical diameter. The same condition of the dura was present in the whole area. A small opening was then made in the dura, and the blood-tinged cerebrospinal fluid spurted out to a distance of about 3 or 4 inches, thus showing the pressure under which it existed. The dura was then cut away over the whole surface from which the bone had been removed, exposing the brain beneath. The brain did not pulsate. The small superficial blood vessels were dilated to three or four times their natural size, and the blood in them was of a dark, venous color.

There was no actual trauma of either the vessels or the brain tissue itself apparent.

In the course of three or four minutes the pulsation in the brain gradually returned; the blood vessels became much less prominent, and the blood in them became of an arterial color. Coincident with these changes, the condition of the patient's pulse and respiration was closely watched, but it could not be determined that any change took place, the pulse rate remaining from 110 to 120. The temporal muscle, which had been retracted anteroposteriorly during the manipulations in the skull and brain, was now allowed to fall together again and was tacked with three or four catgut sutures. The temporal fascia and skin were carefully sutured along the curved section with catgut, a small drain being left down to the brain tissue. A copious dry dressing was applied to the wound. The child recovered from his anesthesia without incident, and in the course of three or four hours was entirely conscious with practically no symptoms of cerebral irritation. He gave the details of his injury, and told his name and address. His convalescence was uneventful; the wound healed per primam, and the pulsation beneath the temporal covering of the brain has been present ever since. There is no tendency for any increase in the size of the cerebral protrusion, but on the other hand, a decrease. Immediately after the operation, and during the following days, it was as much as one-half to three-quarters of an inch above the skull level. It has gradually lessened, until now its maximum is only one-quarter of an inch, and palpation shows it to be less tense than it was two weeks ago. It would have been better to have made an osteoplastic flap, but no instrument for this was at hand, excepting the gouge and mallet, which, under the existing conditions, would have been exceedingly dangerous from the continued jarring necessitated. An attempt to leave the dura in situ and resuture it failed because of the great tension, and the subsequent gradual subsidence of this tension shows that a replacement of this dense membrane would have continued to an excessive intracranial compression, and defeated the very object of the operation. The uncovered brain is a " silent area " and it may be hoped that the pericranium will develop enough thickness and firmness to protect it.

The conditions which determined operation on this boy were, rapidly increasing coma and cerebral excitability, with the strong belief by those observing him, that he was developing the very condition found, namely, cerebral œdema, due to changes in the blood vessels, which would inevitably prove fatal if not relieved. The absence of localizing symptoms left no other course than to produce a decompression of the brain, and thus combat the increasing compression due to loss of tone in the cerebral vessels.

Subsequent note, Feb. 10th, 1908.—The protrusion of the brain has entirely subsided, and given place to a depression one-half inch below the scalp, that is, to its normal level. Except for the skull opening it is entirely normal. The satisfactory result here obtained by no means warrants the conclusion that such lesions invariably produce a picture so easily interpreted. It was the apparent hopelessness of not operating, that acted as the determining factor in deciding for operation.

Cannon and Cushing have studied this condition and both come to the conclusion that an œdema of the brain may arise in this way, through osmotic forces, sufficient to give pressure symptoms by driving out the blood from the medullary centres. In this particular case, this phase had just been reached, and it was apparently rapidly increasing.

CASE VII.—*Diffuse trauma of head. Symptoms of marked cerebral irritation and compression after three hours. More marked over right precentral area. No improvement for three days. Decompressing operation over right motor area. No localized lesion of any moment. Promp improvement and ultimate recovery.*

A woman, age 28 years, fell from a street car on Aug. 24, 1907, striking her head on the pavement. She apparently received no severe injury and walked home. When seen by the ambulance surgeon shortly after, she was quite rational and apparently not much hurt. She refused to go to the hospital. Three hours later the ambulance was again called, and she was brought to the hospital, where examination showed the following condition: Patient lies on right side with knees drawn up. Arms folded across the chest. There is an apparent condition of chilliness, and the bed clothes are drawn up beneath the chin. She assumes above attitude whenever moved. Eyes are closed, breathing natural and she appears in natural sleep unless disturbed. She resents any interference, resisting more or less violently. She moves all extremities freely. Left arm decidedly weaker than right, also left leg weaker than right. Irritation about the head makes apparent a paralysis of lower left facial branches. Pulse 56, hypertension. Left knee-jerk increased. No ankle clonus. Superficial reflexes unsatisfactory. Left pupil reacts to light normally. Right also. Pupils equal, and normal in size. Left eye fixed in external strabismus. Right eye moves on irritation. Palpation of skull shows profuse hematoma all over calvarium, but more on right than left side. For the following three days there was no marked change in any direction, though the slight left-sided paralysis seemed to be somewhat decreased. On Aug. 27th, operation was decided upon because of the signs of continued cerebral pressure as seen in the slow pulse, the irritable condition and the tendency to coma when left undisturbed.

Operation.—Skin incision over right Rolandic area after fissures had been mapped out on the skull. No fractures present. Trephine opening made and enlarged with ronguers. Brain showed increased intracranial pressure, but pulsated slightly. Opening of dura showed slight trace of dark colored blood clot. Reaction with a battery showed that left face area was exposed. Dura sutured with catgut. Rubber tissue drain. Skin flap sutured with interrupted silk sutures. Dressing applied. Patient returned to bed in fair condition with head elevated.

Post-operative notes, Aug. 28th.—The general condition is about the same,but the pulse has risen to 70 and 80, whereas before it ranged from 50 to 60 as the highest. Aug. 28th—The general condition shows improvement, the irritability having almost disappeared. The subsequent course was toward uneventful recovery and by the end of the week the patient was in a normal condition, and the wound was entirely healed. Undoubtedly this case would have recovered without operation. The recovery would have, however, been slower, and there was present a very good chance of later manifestations, the "cerebrasthenia" of Bailey, developing.

A decompressing operation as practised in these two instances therefore seems justifiable in the border-line cases, where no localizing diagnosis can be made. As already pointed out, the same group of symptoms may arise from such divergent causes, and the pathological conditions present be so complex, including all the grades above enumerated under class four, that their relief is problematical. The abdominal surgeon is often confronted with the impossibility of making a diagnosis in obscure lesions, but he has the advantage of being able to deal with whatever may be found on direct inspection of the organs. The brain surgeon cannot do this, and therefore an accurate diagnosis before operation is imperatively demanded despite the difficulties. The deductions to be drawn from these considerations are apparent. Every available point must be weighed in making a diagnosis and a prognosis.

The diagnostic data may be grouped as follows: First—those of the functional disturbance of the brain as a whole. Second—those of the actual damage of brain tissue over local areas. Third—those of the derangement of function due to lesions outside of the brain. Fourth—manifestations of injury shown in organs other than the sensorimotor system. The confusing symptoms falling under the first group are usually present to a greater or less degree, that is, concussion masks and distorts other symptomatic findings. The pupils, for example, may be equally dilated, or one may be inactive while the other may be normal. Statistics should theoretically throw some light on the location of a lesion producing such changes, but a study of the various authorities, and one's own experience, give such divergent results, that little value can be placed on them. Hutchinson has pointed out that the rule is to find the pupil on the injured side dilated, due to a paralysis of the third nerve or its connections. This is more applicable to basal hemorrhage with fracture of the base, however, or with a low hemorrhage from the middle meningeal artery. The explanation of the many exceptions is probably found in the complicating disturbance of function as a whole. The other reflexes are also of uncertain value, and for the same reason. The study of the second group, namely, diagnostic data derived from damage to brain tissue over a localized area, holds out more promise. But even here, casual observation is very deceptive, and only a most rigid analysis of each symptom can lead to any safe conclusion.

Cases II to V illustrate the difficulties encountered here. Sensori motor paralyses are practically the only guides of importance, because the interpretation of pure sensation by patients suffering from head injury is often unobtainable at all, and is always confused. Valuable diagnostic information may be obtained by the presence of symptoms due to associated injuries outside the brain. Of these, the presence of a fracture of the skull is of especial significance, and if the fracture exists on the vertex the associated injury is often easily determined by inspection at this point. On the other hand, fracture at the base of the skull gives no pathognomonic signs other than those of the fracture itself. The bleeding from the nose and ears or the escape of cerebral fluid, and the subconjunctival eccymosis, are of no value in determining the damage to the nervous structures. Occasionally the paralysis of a cranial nerve at its exit from the skull is present, and thus the line of fracture may be determined but without additional information along the lines under consideration. Anatomical considerations may lead to the satisfactory localization of a compressing hemorrhage in such cases, and Cushing earnestly advocates a low exploration in these cases for the purpose, first, of removing what clot can be reached, and second, to give relief to compression by opening the skull. This procedure seems founded on sound principles of brain surgery, and certainly is worthy of wider application than it has had in the past. Under the fourth diagnostic grouping, that is, findings outside of the brain, there are four of value: First—that resulting from a tapping of the spinal cord. Second—a protrusion of the eyeballs. Third—the condition of the choked disc due to increased intracranial pressure. Fourth—the changes in the circulation and respiration due to interference with their medullary centres. The presence of blood in the spinal fluid is often the only diagnostic proof that the brain, or at least the dura, has suffered gross damage. This blood can have two sources, one from ventricular hemorrhage, and the other from subdural hemorrhage. Hemorrhage within the brain tissue does not give it. Fracture of the base usually does.

From this sign we may correctly infer a lesion other than severe concussion when this question is in doubt. The protrusion of the eyeball also shows increased intracranial pressure and is absent in concussion alone. The same is true of the choked disc, which is more marked on the side of greater pressure. This sign is of the utmost importance and often turns the scale in making the diagnosis of a compression being present. Cushing has shown that it is a very delicate sign and changes rapidly with the change in pressure.

The changes in the circulation and the respiration are equally important. Every operating room where brain surgery is done should be equipped with the means of accurate determination of these functions. The rate and force of the pulse are most important guides. A slow rate with marked variations during short intervals is significant of various stimulation with a tending toward paralysis of that centre. Many observers have found the change in blood pressure to be in direct relation to the change in intracranial pressure, but Cushing was the first in this country to emphasize that the rise in the former was a conservative act to keep up the circulation through the compressed centres. He reports a case where the blood pressure rose above 300 mm. of mercury, with a prompt fall when the intracranial pressure was released.

Eyster has worked out the changes in respiration. He showed that the arythmic respiration is due to an alternate anæmia and vascularization of the centre with the failure or success of the blood pressure to keep up with the intracranial pressure. He also showed that the irritability of the respiratory centre is lost from anæmia sooner than the other centres. A rising blood pressure, with arythmic respirations, therefore betokens the last stage of compression as given by Kocher. Hence, a close watch on these functions is most important for diagnosis and prognosis.

From the above outline of the complicated conditions found in brain trauma, one must conclude that it is among the most difficult pathological conditions the surgeon is called on to treat. But few words are needed to consider the operative procedure to be followed, once operation has been decided upon. Practically all parts of the skull, except the base and the lower occipital region, may now be opened by means of the osteoplastic flap. It is outside the scope of the present paper to discuss the technique and the details of this method in its manifold applications. It may, however, be said that the use of the trephine and the rongeur has largely been discarded for one form or another of the electrically run burr and saw, and that with the latter, very large areas of the brain may be exposed with the minimum of shock and damage to the skull. We may even indulge in the hope that the future will see still further improvements in these implements, to such an extent that the exploratory surgery of the brain may be more rational and less hap-hazard than it has been in the past. It is safe to say, that along these lines lies the development of a technique which will yield results far more favorable than any heretofore seen.

The seven cases here reported have been selected as illustrations of groups three and four of the original classification, adapted for the therapeutical consideration of intracranial lesions. Case I is a typical example of the definitely localized lesion, giving almost unmistakable symptoms. Case II shows a similar lesion, but with somewhat modified symptoms. Cases III, IV and V illustrate the sources of error where the clinical picture simulates a definitely localized lesion, but where no such lesion exists. Cases VI and VII were selected as examples of the fourth group or the border-line cases. In neither could a localized lesion be diagnosed, nor was one found at operation. Yet the general picture seemed to justify something being done to relieve cerebral irritation and pressure. The decompressing operation fulfilled these indications, and proved of value in both cases.

A critical analysis of the subject as illustrated in the seven cases warrants the following conclusions: First—In but rare cases, namely, those of isolated injury affecting the sensorimotor area, can a positive focal diagnosis be made. Second—All grades of brain injury may be found in different parts of the same brain. Third—A general concussion may be followed by secondary changes in the circulation which, if not relieved, produce pressure and death. Fourth—A pure decompressing operation is indicated in two conditions: (*a*) for the relief of pressure due to inaccessible hemorrhage, and (*b*) to relieve the pressure arising from traumatic œdema of the brain. Fifth—Operation done without a very definite object in view, which object is based on careful diagnosis, is apt to be more harmful than helpful. Sixth—the whole subject is fraught with manifold difficulties and the brain surgeon should strive to become a practical neurologist in organic lesions.